Nutritional Support In The Critically Ill Child

Col Uma Raju^{*}, Surg Lt Cdr Sanjay Choudhary⁺, Lt Col MM Harjai[#]

MJAFI 2005; 61 : 45-50

Key Words : Malnutrition; Critical care; Enteral nutrition; Parenteral nutrition

Introduction

Malnutrition in hospitalized patient is increasingly being recognized as an important factor determining outcome of the disease. There is growing evidence that early and appropriate goal oriented nutritional support in the ill child aids recovery [1,2]. Current nutritional management is based on a rapidly emerging knowledge of the special nutritional requirements related to the vastly different physiologic and metabolic characteristics of these patients. These children often present with significant metabolic derangements in protein and energy metabolism characterized by increased protein breakdown unsuppressed by protein or energy intake, reprioritization of protein synthesis with increased synthesis of acute phase proteins, decreased synthesis of structural proteins and increased turn over. In addition, there is also glucose and lipid intolerance [3]. Adequate nutritional and metabolic support of the critically ill child under these conditions is a daunting task [4]. It involves an accurate calculation of caloric delivery with a precise mixture of carbohydrates, proteins, lipids and micronutrients, which needs periodic review. The route of nutrient delivery also needs contemplation with increasing popularity of enteral feeding in this group [5]. Emerging knowledge of the non-nutritional functions of nutrients has added another dimension to critical care nutritional support.

Pathogenesis

Systemic stress response in the critically ill child is a hypersympathetic one. At the macro level, gut motility especially of the stomach and small intestines is reduced. Absorption of nutrients and drugs may be erratic secondary to villous atrophy, associated with altered motility secondary to ischaemia and necrosis. Consequently increased bacterial and their toxin translocation occur, which may suppress normal immune mechanisms and promote the activation of cytokine

synthesis in the liver. Multi organ derangements, particularly of the liver and kidneys affect not only nutrient but also drug metabolism.

At the cellular level, macrophages and polymorpholeukocytes release various peptides like cytokines, interleukin 1 and tumor necrosis factor [6,7]. The stress response is associated with elevated catecholamines, aldosterone, antidiuretic hormone, glucocorticoids, insulin and glucagons [8]. Despite elevated insulin levels, hyperglycaemia and glucose intolerance are frequently observed because of counter regulatory hormones primarily due to mobilization of alanine, glutamine and other amino acids from muscle and their biosynthesis to glucose and urea by the liver. There is insuppressible lipolysis and reduced ketogenesis. Most critically ill children are in negative nitrogen balance as protein catabolism far exceeds synthesis. The net effect is rapid depletion of lean body mass termed as autocannibalisation. Neonates and children are particularly susceptible to this. Intrinsic lack of endogenous stores and greater baseline requirements compound problems. An appropriately designed mixed fuel system of nutritional support replete in protein does not quell this metabolic response but can result in anabolism and continued growth in ill children [9].

Critical illness is associated with increased energy demands related to stress. In order to ensure that optimal calories are provided to the sick child, while calculating energy requirements, the basal energy expenditure (Kcals) should be multiplied by a stress factor, which is specific for that particular ailment. This pattern of requirement will vary according to the disease and from patient to patient and needs to be considered while formulating individual diets, which must be modified from time to time.

General Principles

Until recently, critically ill children received nutritional

^{*}Senior Adviser (Paediatrics & Neonatology), Command Hospital (Southern Command), Pune-40, ⁺Graded Specialist(Paediatrics), 166 Military Hospital, C/o 56 APO, [#]Classified Specialist (Surgery & Paediatric Surgery), Army Hospital (R & R), Delhi Cantt

support based on the predicted energy expenditure (PEE) extrapolated from normograms of healthy children. The aim was to maximize caloric delivery in the hypermetabolic and stressed state the baby was passing through. On studying the energy requirements in these children by indirect calorimetry, it was observed that the PEE was grossly overestimating their needs and thereby the excess calories provided added further to the metabolic derangements. Therefore, the current concept is to provide hypocaloric nutritional support during the early unstable Ebb phase followed by eucaloric and even hypercaloric feeding depending upon the stress factor during the recovery flow phase [10,11]. As a broad outline this amounts to 20-30 calories / kg / day during the Ebb phase followed by 50-100 calories / kg / day during the recovery phase [12,13].

Assessment of nutritional status

Nutrition assessment is an integral part of evaluation of the critically ill child. Its goal is to identify malnourished children and those who are at risk of developing it. Malnutrition is known to affect wound healing, infection rate, mortality and morbidity making early identification of children at risk essential [14]. Designing effective nutrition regimens for the critically ill requires an understanding of the energy needs of each patient. Many disease processes result in elevated caloric requirements whereas some clinical procedures and medications may diminish the metabolic response [15,16].

Assessment consists of a detailed history taking and clinical examination, which is evaluated in conjunction with anthropometry and appropriate lab investigations as shown in Table 1. Nutrition requirements may also be determined by measurement from complex formulas which provide useful guidelines on caloric management in the critically ill. Some of these are:-

- (a) Basal Metabolic Rate (BMR) : There are certain basic differences in basal energy expenditure between children and adults. These are a relatively higher BMR, considerably lesser nutritional stores, growth and the fact that metabolic organs make up

Table 1

Clinical nutritional assessment of the ill child

Condition	Measurement	Deficiency state
Acute PEM	Weight/50 centile weight for length	< 0.8
<i>Macronutrients</i>		
Fat	Triceps skin fold thickness	< 5th centile
Somatic proteins	Mild arm circumference	< 5th centile
Visceral proteins	Serum albumin	< 2.5 gm%
<i>Micronutrients</i>		
Immune state	Individual assay	
	Total lymphocyte counts	< 1000/cmm
	Skin anergy tests	negative

a greater percentage of body weight in children. Normograms are available for varying age groups one of which is shown in Table 2. Harris Benedict formula has been the time honored method of estimating basal energy requirement in Kcals / day viz:-

$$\text{Males} = 66 + 13.7W + 5H - 6.8A, \text{Female} = 65.5 + 9.6W + 1.7H - 4.7A$$

W=weight in kgs, H=height in cms, A=age in years.

- (b) Indirect Calorimetry (Metabolic Cart) is the most popular method of assessing caloric requirements in the Paediatric Intensive Care Unit (PICU) in the western world. It is based on the assumption that the oxygen consumed and carbon dioxide produced by the body is equal to the energy expended in metabolic processes [17].

$$\text{Energy Expended (EE)} = 3.586\text{VO}_2 + 1.443\text{VCO}_2 - 21.5$$

VO₂ = Oxygen consumed, VCO₂ = Carbon dioxide produced.

The main shortfall is that in the critically ill, metabolism varies considerably necessitating frequent review.

- (c) Respiratory Quotient: VCO₂ / VO₂ below 0.7 suggests that the main fuel is fat and when > 1.0 suggests lipogenesis.
- (d) Stress Factor is a simple method of estimating energy requirements in the hospital setting described by Souba and Wilmore [18]. The energy expended is calculated by multiplying the BMR with 1.25 and the stress factor for the particular illness. The stress factor in various illnesses as assessed by Souba and Wilmore is shown in Table 3.

Whilst these methods form general guidelines, one

Table 2

Basal metabolic rate - normogram in Kcals/day

Age (years)	Male	Female
1 - 3	51.3 - 53.0	51.2 - 53.0
4 - 7	47.3 - 50.3	45.4 - 49.9
8 - 11	43.0 - 46.5	39.2 - 41.3

Table 3

Stress factor (Souba & Wilmore)

Condition	Stress factor
Mild starvation	0.85 - 1.00
Postoperative state	1.00 - 1.05
Cancer	1.10 - 1.45
Peritonitis and sepsis	1.05 - 1.25
Multi trauma, burns	1.20 - 1.55

$$\text{Energy expended} = \text{BMR} \times 1.35 \times \text{stress factor}$$

should administer enough calories to achieve a clinical improvement with adequate weight gain, positive nitrogen balance as indicated by muscle mass as well as measurement of skin fold thickness to assess subcutaneous fat [19].

Nutrients needed by the body may be broadly classified functionally as:-

- (a) Body builders – proteins; (b) Energy producers – carbohydrates and fats; (c) Protective nutrients – vitamins, minerals and trace elements

The main purposes of nutrition are growth and restoration of body proteins, the meeting of energy demands of metabolic processes being fulfilled mostly by carbohydrates and fats. In a balanced diet, the proteins, carbohydrates and fats are provided in the ratio of 15:50:35. For optimal utilization of proteins, at least 150 non-protein calories need to be administered per gram of nitrogen provided. In certain catabolic situations, this requirement may increase to 250-300 non protein calories / gm nitrogen provided. These requirements vary with the age of the patient, severity of disease and pre existing illness [20]. Broad guidelines of the energy and macronutrient requirements in the various age groups in children are as shown in Table 4.

The route of nutrient delivery influences the total caloric requirement. It has been observed that to achieve comparable accretion rates, 75-80% of the enteral requirements need to be provided parenterally.

Table 4
Nutritional requirements /kg/day - Paediatric patients

Age (years)	Energy (Kcals)	Proteins (gms)	Carbohydrates (gms)	Fats
Neonate	100 - 150	2.5 - 3.0	15 - 20	3
< 1	100 - 120	2.0 - 2.5	12 - 15	3
1 - 6	75 - 90	1.5 - 2.0	9 - 12	2 - 3
7 - 12	60 - 75	1.0 - 1.5	7 - 9	2

Proteins: The usual protein requirement in the critically ill child ranges from 1.5-3 gms / kg /day. This takes into account the normal maintenance requirements, metabolic demands of disease and replacement of abnormal losses. Proteins can be provided enterally as short chain peptides or complex proteins. Intravenous alimentation entails the administration of crystalline aminoacid solution with an adequate balance of essential / non-essential aminoacids. Some of the paediatric intravenous aminoacid solutions available in the Indian market are shown in Table 5. Attention should be paid to the percentage of branched chain aminoacids in the infusate, which are particularly useful in liver failure and in multi trauma. Protein restriction however is indicated in renal failure.

Table 5
Paediatric parenteral aminoacid preparations

Brand	Company	Strength
Aminoplasmol	B Braun	5 gm%
Alamin N	Albert David	6 gm%
Aminoven	Frenesius	6 gm%
Aminocore	Clarix	5 gm%
Celemin	Clarix	5 gm%

Carbohydrates: The goal of carbohydrate administration is to provide energy and to spare lean body mass maximally. It usually accounts for 50-60% of the non-protein calories to be administered. It may be provided enterally as complex carbohydrates or oligosaccharides with lactose free diet being provided to the intolerant patient. Parenterally, glucose is the sugar of choice as it is an inexpensive, naturally occurring substrate. The daily requirement ranges from 5-25 gms/ kg. During the initial critical phase, glucose infusion should not exceed 4-6 mg/kg/minute and should be titrated with blood glucose levels. The critically ill patient may exhibit glucose intolerance and hence regular serum glucose levels, weight gain and nitrogen balance need to be monitored to decide optimal dosage. Over administration leads to hyperthermia, increased CO₂ production and hepatic steatosis. Hence hyperglycemia should be treated with reduction in percentage of dextrose infusion when blood glucose levels exceed 200 mgm% and with insulin infusion at the rate of 0.05-0.1 IU/kg/hr when levels exceed 250 mgm%.

Lipids: Approximately 15-30% of the calories should be provided by lipids which have a nitrogen sparing effect. They may be provided orally as medium chain triglycerides. Vegetable fats are better tolerated in the ill patient. Small doses of safflower / soya / coconut oil (0.5 gm/kg/day) not only provide calories but also medium chain triglycerides which are easily absorbed. Omega 3 fatty acids contained in fish oils not only provide lipid calories but are also an invaluable immunonutrient.

Fats appear to be more suitable as an energy source as compared to carbohydrates as lipid preparations provide high caloric density in comparatively low volumes along with an ideal R/Q ratio suitable for most sick patients who have borderline respiratory reserve and may have excess body water. For intravenous alimentation lipids are provided as triglycerides stabilized with egg phospholipid. However, most of the commercially available preparations contain more phospholipids than required to emulsify triglycerides, the excess phospholipids being converted into liposomes. The available 10% lipid solution generally provides two to four times the amount of liposomes as compared to

the 20% lipid solution. It is speculated that the excess liposomes compete with the triglyceride rich particles for lipase binding sites. Hence the liposome rich fat emulsions are poorly tolerated with an increased incidence of hypertriglyceridemia and hypercholesteremia. Therefore 20% lipid emulsions with reduced phospholipid content has been found to be better tolerated than the 10% solution. In the recent past, 10% lipid solutions with reduced phospholipid content are available which has resulted in better tolerance.

Lipid therapy should be started in dose of 0.5 gm/kg/day and increased in increments of 0.5 gms/kg/day, not exceeding 4 gms/kg/day. Monitoring of lipid profile is essential as lipid intolerance in the critically ill child is known. Other side effects include fat pulmonary micro embolism, thrombocytopenia and reduced leukocyte functions. With the current understanding of long chain triglycerides being potentially immunosuppressive, alternate lipid sources with medium chain triglycerides and W-3 fatty acids are being increasingly used [21].

Electrolytes and Minerals: The critically ill child should be provided not only maintenance requirements of electrolytes but ongoing losses particularly from GIT need to be replenished. Phosphorus required due to accretion of calcium and phosphorus in the bone due to protein anabolism carries a requirement of 1.3 mmol/kg/day for every 400 mgm nitrogen/kg and 0.8 mmol calcium/kg. Magnesium requirements are usually met by 0.4 mmol/kg/day [22].

Vitamins must be provided to meet maintenance requirements as well as cater to losses and supervening stress. When provided intravenously, vitamins especially A, C, riboflavin and pyridoxine may be lost by adherence to plastic tubing and by photo degradation. Therefore vitamins should be added to parenteral infusates shortly before administration and should be protected from light [23].

Nutraceuticals / Immunonutrients: Of recent interest is the emergence of the non-nutritional potential of certain nutrients. These include glutamine which when supplemented in the critically ill child is believed to improve nitrogen balance and gut function and also to reduce infection rate [24]. Ornithine produces glutamine in vivo, increases growth hormone activity and has also been found to reduce the incidence of metabolic acidosis. Arginine, fiber, omega 3 fatty acids, vitamins (A,C and E) and certain trace elements viz selenium, copper and zinc are believed to improve immune functions. Omega 3 fatty acids, dietary peptides and nucleic acids aid fat metabolism in the critically ill child. They are believed to play an active role in enhancing immunity and help wound healing. Trials on immunonutrition have shown beneficial results [25].

Routes of Nutrient Delivery

Enteral: Early enteral feeding in the critically ill child has been found to avert ulcerative complications, preserve indigenous intestinal flora, prevent mucosal atrophy, maintain the enterohepatic enzyme system and reduce the incidence of sepsis and cholestatic jaundice [26,27]. Feeds can be provided orally or in case of gastric atonicity, transpyloric. These may be provided as intermittent, bolus or continuous infusions depending on tolerance. Drugs such as metchlorpromide, cisapride and erythromycin when provided as primers have reduced the incidence of gastric atonicity and have also aided transpyloric tube negotiation [28]. If cisapride is provided, ECG monitoring to exclude QT segment abnormalities and drug interactions particularly with azoles, macrolides and protease inhibitors need to be kept in mind. Diets include low molecular weight elemental formulas, polymeric or custom made modular formulas. Some of the commercially available, ready to use enteral diets for paediatric patients are shown in Table 6. Side effects are few and relate to aspiration, diarrhea, abdominal distension and those related to the tube [29].

Table 6
Enteral nutrition commercial formulas

Product	Company	Calories/100 gms
Nourish	Clarix	518
Simyl MCT	FDC	460
Pedia Sure	Abbott	496
Prosoyal	FDC	506
Impact (IB)	Novartis	484
Novasure	Novartis	400

In sick neonates on parenteral alimentation, minimal enteral feeding providing 4-20 kcals/kg/day has been found to be beneficial. This form of nutritional therapy has been associated with better weight gain, greater decline in serum bilirubin levels, less cholestasis, rapid maturation of the gut, increased feed tolerance and quicker recovery.

Parenteral Nutrition: If the GIT cannot be used adequately, parenteral nutrition becomes necessary. It can be provided by peripheral venous access unless prolonged IV alimentation is contemplated when a central line needs to be established. This allows the use of concentrated infusates.

Parenteral nutrition is initiated with a glucose infusion at a dosage of 5 mgm/kg/mt and increased in daily increments to a maximum of 25 gm/kg/day. Aminoacids are added on the 2nd day starting at 0.5 gm/kg/day and gradually increased by 0.5 gm/kg/day to a maximum of 3 gm/kg/day. Lipids are added on the 3rd day at 0.5-1 gm/kg/day and increased by increments of 0.5 gm/kg/day to maximum of 4 gm/kg/day. The fluids should be

customized and prepared on a daily basis as per requirement and tolerance. The calculated quantities of aminoacids, glucose and electrolytes are mixed in a bottle to which calcium gluconate, phosphates, multivitamin and heparin is added. The nutrient mixing should be carried out under aseptic conditions preferably under a laminar flow workstation. A bacterial filter should be attached to the IV line to further limit microbial contamination. Lipids should preferably be administered by a separate line, the two being coupled by a three-way stopcock prior to entry into the vein. In the recent past single unit mixtures with all of the required nutrients including lipids are being provided in some centres for ease of administration. The serious disadvantage of such a system is that a bacterial filter cannot be utilised as the lipids cannot transgress the filter leading to a blockage. In order to provide successful parenteral nutrition, clinical and biochemical monitoring is essential. Clinical monitoring includes vital parameters recording, checking IV sites and rates, daily weight record and maintaining an intake output chart meticulously. Biochemical monitoring entails measurements of blood electrolytes, sugar, creatinine, calcium, phosphates, lipid profile and serum protein levels every third day during the initial unstable phase and weekly and SOS thereafter. Urine should be checked for sugar and ketone bodies three to four times daily. It is essential to use micro methods for biochemical monitoring and the blood loss due to sampling must be replenished to prevent anemia. Complications relating to metabolic derangement, infection and mechanical line problems should be looked for and judiciously attended to. It must be realized that successful parenteral nutrition requires constant vigilance, dedicated personnel and specialized facilities in order to achieve maximum benefits and minimum complications. However this form of therapy is neither as nebulous nor as difficult as it may appear, and its benefit should therefore not be denied to the patient who needs it most.

Nutrition in special situations: In burns, sepsis and trauma formulas rich in branched chain aminoacids upto 35-50% of aminoacids administered need to be provided. In liver failure, increased branched chain aminoacids and lower concentration of aromatic amines should be used. In addition fiber, lactulose and lactitol are effective [30]. In renal failure, a high concentration of essential aminoacids with a greater non-protein caloric concentration needs to be provided.

Conclusion

Nutritional management is an integral and vital part of criticare support. The stress of severe illness complicates the delivery of adequate nutrients. Enteral feeding has several advantages. However there are

specific instances when parenteral nutrition as an adjunctive or as sole therapy becomes mandatory to meet nutritional needs. Whatever the modality, with meticulous attention to nutrient requirements along with rigorous monitoring, it is possible to provide full nutritive support to the critically ill child.

References

1. Kinn S, Scott J. Nutritional awareness of critically ill surgical high dependency patients. *Br J Nurs* 2001 Jun;10(11):704-9.
2. Iyer PU. Nutritional support in the critically ill child. *Indian J Pediatr* May;69(5):405-10.
3. Coss Bu JA, Klish WJ, Walding D, Stein F, Smith EO, Jefferson LS. Energy metabolism, nitrogen balance and substrate utilization in critically ill children. *Am J Clin Nutr* 2001 Nov;74(5):664-9.
4. Curley MA, Castillo L. Nutrition and shock in the critically ill patient. *New Horizon* 1998 May;6(2):212-25.
5. Briassoulis G, Zavras N, Hatzis T. Malnutrition, nutritional indices and early feeding in critically ill children. *Nutrition* 2001 Jul-Aug;17(7-8):548-57.
6. Dinarella CA. Biology of interleukin 1. *FASEB J* 1998;9:445-52.
7. Le J, Vilcek J. Tumor necrosis factor and interleukin 1: cytokines with multiple overlapping biological activities. *Lab Invest* 1987;56:234-45.
8. Goldstein SA, Elwyn DH. The effects of injury and sepsis on fuel utilization. *Ann Rev Nutr* 1989;9:445-9.
9. Shaw SB, Jalesic T. The metabolic needs of critically ill children and neonates *Semin Pediatr Surg* 1999 Aug;8(3):131-9.
10. Briassoulis G, Venkataraman S, Thompson AE. Energy expenditure in critically ill children. *Crit Care Med* 2000 Apr;28(4):1166-72.
11. Steinhorn DM. Energy expenditure (REE) in critically ill children is lower than expected. *Pediatr Res* 1993;41 A:33-8.
12. McCrowen KC, Friel C, Sternberg J. Hypocaloric total parenteral nutrition: effectiveness in prevention of hyperglycemia and infectious complications. *Crit Care Med* 2000;28(11):3606-11.
13. Patino JF, De Pimento, Vergara A. Hypo caloric support in critically ill. *World J Surg* 1999;23(7):795-806.
14. Bettle J, Roberts KE. Nutrition assessment of the critically ill child. *AACN Clin Issues* 2000 Nov;11(4):498-506.
15. Fung EB. Estimating energy expenditure in critically ill adults and children. *AACN Clin Issues* 2000 Nov;11(4):480-97.
16. Pollack MM. Nutritional failure and support in pediatric intensive care. In Shoemaker WC, Thompson WC, Holbrook PR editors. *Textbook of critical care* – Philadelphia, WB Saunders 1984;694-769.
17. Manthous C. In Hall JB, Schmidt GA, Wood LDH. editors. *Principles of critical care*. McGraw Hill Inc, Singapore 1993;12:79-86.
18. Souba WW, Wilmore DW. Planning TPN. *Clin Anaesthesiol* 1983;1:633.
19. Khilnani P, Singh R, Uttam R. nutritional support of the critically ill child. *Indian J Practical Pediatr* 2000 Dec;2(4):348-56.

20. Heird WC. Aminoacid and energy needs of pediatric patients requiring parenteral nutrition. *Pediatr Clin North Am* 1995;765.
21. Olivier Goulet, Sophie de Potter, Helena Antebi, et al. Long term efficacy and safety of a new olive oil bases IV fat emulsion in paediatric patients: a double blind randomized study. *Am J Clin Nutr* 1999;70(3):338-45.
22. Ricour C. Total parenteral nutrition in children. *Annales Nestle* 1988;42(2):61-72.
23. Kalhan SC, Price PT. Nutrition and selected disorders of gastrointestinal tract. In care of the high risk neonate. Klaus MH, Fanaroff AA editors WB Saunders Philadelphia 2001;5th ed,147-98.
24. Barbul A. immunonutrition comes of age. *Crit Care Med* 2000;28:884-5.
25. Barbosa E, Moreira EA, Goes JE, Faintuch J. Pilot study with a glutamine supplemented enteral formula in critically ill infants. *Rev Hosp Clin Fac Med Sao Paulo* 1999 Jan-Feb;54(11):21-4.
26. Atbenson S, Scaffert F, Bihari D. A prospective, randomized, double blind controlled clinical trial of enteral immunonutrition in the critically ill. *Crit Care Med* 1998;26:1164-72.
27. Irving SY, Simone SD, Hicks FW, Virger JT. Nutrition for the critically ill child: enteral and parenteral support. *AACN Clin Issues* 2000 Nov;11(4):541-8.
28. MacClaren R, Kuhl DA, Diakerson RN. Sequential single doses of cisapride, erythromycin and metchlorpomide in critically ill patients intolerant to enteral nutrition. A randomized placebo controlled crossover study. *Crit Care Med* 1999;279(12):2799-805.
29. Reed MD. Principles of enteral nutrition. In: A practical guide to Pediatric intensive care. 3rd ed. Editors Blumer JL, Mosby Yearbook 1990;592-610.
30. Muller KD, Waber FC. Role of nutrition in hepatic encephalopathy. *Semin Liver Dis* 1991;11:292.

Quiz

Radiological Quiz

Surg Cdr HS Nagar*, Surg Cdr PC Hande⁺

MJAFI 2005; 61 : 50

A newborn presented with severe respiratory distress after the first feed. On examination, he had apparent dextrocardia, absent air entry on left side of chest and a scaphoid abdomen. These were the findings in chest X-ray.

1. What is your diagnosis?
2. What is the closest differential diagnosis?
3. What is the cause of this pathology?
4. What immediate action is required?

Fig. 1 : Plain x-ray chest and abdomen

Answer to the Radiological Quiz - pp 78

*Classified Specialist (Surgery & Paediatric Surgery), INHS Kasturi, Lonavala, ⁺Classified Specialist (Radiology), INHS Asvini, Mumbai

Received : 15.07.2003; Accepted : 20.01.2004